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THE EFFECT OF INSULATING BLOOD WARMER OUTPUT TUBING ON THE TEMPERATURE OF PACKED RED BLOOD CELLS AT LOW FLOW RATES

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in Nurse Anesthesia at Virginia Commonwealth Universtity

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Abstract

THE EFFECT OF INSULATING BLOOD WARMER OUTPUT TUBING ON THE TEMPERATURE OF PACKED RED BLOOD CELLS AT LOW FLOW RATES

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A true experimental research design was selected to determine the effect of insulating blood warmer output tubing on the temperature of packed red blood cells at the point of patient entry at low flow rates. Utilizing a surgical suite, every attempt was made to replicate a true surgical setting. Flow control pumps were used to regulate flow rates of 300, 600, and 900 milliliters per hour (ml/hr) through Fenwall Laboratories blood warming coils. The warming coils were immersed in a Hemokinatherm® warm water bath and the pre-attached output tubing extension, 69 inches in length, was attached to the head of the operating room table. Temperature was measured in °C at the following sites (a) the room air, (b) the point just prior to entering the blood warmer, (c) the point immediately after exiting the blood warmer, and (d) the end of the pre-attached output

tubing extension. Room temperature was maintained at 20.0 ± 0.5 °C. Insulated and non-insulated blood warmer output tubing was compared. Results of the study showed that insulation significantly decreased the amount of heat loss that occurs in non-insulated output tubing. Increased flow rate was also found to decrease heat loss in both insulated and non-insulated output tubing. Data analysis was performed using an analysis of covariance.

Chapter One

Introduction

With the advent of complicated and lengthy surgeries, hypothermia has become a common and often overlooked condition affecting patients undergoing general anesthesia. Hypothermia is as a thermal imbalance where body core temperatures drop below the normothermic range of 36 to 38 °C (Grayson & Kuehn, 1979). Hypothermia has diverse effects upon the various organ systems within the human body which may compromise the patient's well-being during their peri-operative experience. For the very young, the aged, and the debilitated patient, the risk of developing hypothermia intraoperatively is greatly increased. Factors which may lead to a loss of body heat in the surgical patient include such variables as the cold environment of the operating theater, the use of cold antiseptic and irrigation solutions, the administration of cold intravenous fluids, and the administration of cool/dry anesthetic gases.

It is common practice today to implement methods that decrease heat loss during the peri-operative experience, and

thus, prevent hypothermia and its detrimental effects. Methods commonly utilized intraoperatively include:

1. Increasing the temperature and humidity of the operating theater.
2. Covering the patient with warmed blankets.
3. Utilizing warm water heating blankets.
4. Humidifying and warming of inhaled gases.
5. Warming irrigation and preparation solutions.
6. Warming intravenous fluids and blood products prior to administration.

The warming of blood products prior to intravenous administration has received a great deal of attention, and several researchers advocate this practice as the most effective method to minimize the detrimental effects of hypothermia (Boyan & Howland, 1962; Aldrete, 1985; Fried, Satiani, & Zeeb, 1986). Blood is normally stored at a temperature of 4.0 °C which increases the viability of the red blood cell by decreasing the rate of glycolysis within the cell (Miller & Brzica, 1986). The infusion of one liter of blood at 4.0 °C will produce a decrease in body temperature of 0.5 °C in the average unanesthetized adult (Tollofsrud, 1984). Miller and Brzica (1986) state that this fall in body temperature can be attenuated if the blood is warmed prior to its administration.

Currently, the most common method of warming blood is the warm water bath method. The process involves infusing

blood through tubing immersed in a thermostatically controlled water filled reservoir. Russell (1974) found that these particular warmers could raise blood temperature to 32.0 °C at flow rates up to 150 milliliters per minute (ml/min). A major limitation of the warm water fluid warmer, as reported by Russell (1969), Vaghadia (1986), and Rymers (1988), was increased lengths of output tubing and low flow rates (less than 25 milliliters/minute), resulted in an increase in heat loss through the output line to the patient. As a result, the temperature of the blood at the point of patient entry then approaches ambient air temperature. As Vaghadia (1986) points out, many anesthesia practitioners use warm water bath fluid warmers to heat blood and fluids which are slowly administered to the patient. These slow flow rates allow the fluids to cool in the output tubing, and therefore, no real benefit is derived as a result of utilizing the warmer. Thus, the time and cost of utilizing the warmer results in no actual service to the patient.

The anesthetist should utilize all methods available to maintain the patient's body temperature and prevent hypothermia. The administration of warm fluids and blood products is one method available. The focus of this study was to determine the effectiveness of decreasing heat loss from warmed blood by insulating blood warmer output tubing. Flow rates less than 1,200 milliliters per hour (ml/hr) were

studied because significant heat loss was found to occur with slower flow rates (Russell, 1969; Vaghadia, 1986; Rymers, 1988).

Statement of the Problem

Hypothermia is known to produce cardiac dysrhythmias, an increase in peripheral vascular resistance, a decrease in liver function, and changes in pulmonary function (Martyn, 1981). It is also believed that blood and fluid warming provide one of the most effective means to prevent hypothermia (Boyan & Howland, 1963). However, the prevention of hypothermia and its detrimental effects on the body is essentially ineffectual when significant heat loss occurs in the blood warmer output tubing when infusing fluids at low flow rates. Since the common practice of warming fluids in this manner may be less effective than previously thought, the danger of hypothermia remains a threat to the patient.

Statement of the Purpose

It has been observed by other researchers that heat loss occurs in the blood warmer output tubing at low flow rates (i.e., < 1,200 ml/hr). The purpose of this study then, is to determine whether a statistically significant difference in heat loss occurs between insulated and non-

insulated blood warmer output tubing at flow rates below 1,200 ml/hr.

Hypothesis

There will be no difference in heat loss between insulated and non-insulated blood warmer output tubing at low flow rates.

Variables

Independent. The independent variables are: (a) Fenwall blood warmer output tubing with 0.5 inch thick foam insulation, and (b) infusion rates less than 1,200 ml/hr.

Dependent. The dependent variable is the difference in heat loss between insulated and non-insulated blood warmer output tubing at the point of patient entry.

Theoretical Definitions

For the purpose of this study the following theoretical definitions will be used:

Flow rate. The volume of fluid administered in a given time period and expressed in milliliters per hour (ml/hr).

Temperature. A numerical value on the Celsius scale expressed in degrees Celsius (°C).

Blood warmer. A device that has the ability to heat fluid passing through it. The type used for this study was

a metal heat sink with an electrical coil, located in the base of the device, to heat the water filled reservoir. Fluids being administered to the patient are infused through intravenous tubing immersed in the heated water prior to infusion.

Heat loss. A measurement of differences in temperature between two points (exit point of blood warmer and patient entry point of output tubing).

Infusion pump. A device that delivers a precise amount of fluid per unit time. The type used for this study was electrically powered with a volumetric control.

Operational Definitions

For the purpose of this study the following operational definitions were used:

Output tubing insulation. A 0.5 inch thick, 0.75 inch wide, foam weatherstrip tape non-overlapping and continuously wrapped around the blood warmer output tubing from the point of exit from the water bath to the patient entry point.

Low flow rates. Fluid flow rates less than 1,200 ml/hr.

Assumptions

1. The infusion pumps delivered fluids at the prescribed flow rate.

2. The thermometer was accurate in the measurement of temperature as described by the manufacturer.

Limitations

1. More than one thermometer was used in the study.

2. More than one infusion pump was used in the study.

3. Room temperature may have varied during the period of experimentation.

Delimitations

1. All thermometers were calibrated to the same temperature against a control thermometer.

2. Room temperature was thermostatically controlled to a temperature between 20.0 $\pm$ 0.5 °C.

Conceptual Framework

Temperature Regulation

Man, as a homeothermic animal, must maintain a body temperature within narrow limits in order to survive. Body temperature is dependent upon a precise balance between heat production and heat loss. Temperature regulation in humans involves two types of physiological functions (a) behavioral or voluntary, usually conscious; and (b) autonomic, usually involuntary and/or subconscious (Flacke, W., Flacke, J., Ryan, & Britt, 1983).

Behavioral responses include changes in body posture, locomotion, and provision of "basic necessities" such as clothing and shelter. Flacke et al. (1983) have stated, "behavioral activities are considered the most important response for temperature regulation utilized by man" (p. 277). Of course, these responses are negated under general anesthesia. Involuntary responses involve autonomic functions, such as cutaneous vasoconstriction and non-muscular thermogenesis to decrease heat loss. Non-muscular thermogenesis involve chemical processes to generate heat which are not related to skeletal muscle functions. These processes mainly involve the liver and metabolism of brown fat tissues which are mediated by beta-adrenergic hormones. Brown fat metabolism is a process used by infants but not adults (Hey, 1972). Skin vasodilation and increased sweating are autonomic functions that increase heat loss. Increased muscle tone and shivering are other responses that increase heat production. These are involuntary or subconscious in nature (Flacke et al., 1983).

Temperature regulation, in the human body, is controlled by an area located in the anterior hypothalamus of the brain (Howat, Barker, Vale, & Ellis, 1973). This area, the thermoregulatory control center, senses the temperature of the blood flowing through its structure. In addition, the thermoregulatory control center is supplied with information about the peripheral environment via skin

sensors that respond to changes in temperature. Neural pathways carry information from the skin sensors to the the temperature regulatory control center. Input from skin sensors affect the physiologic responses determined by the thermoregulatory control center. The sensors consist of both warm and cold receptors. The firing rate of these peripheral receptors increase or decrease according to the skin temperature. The thermoregulatory center is capable of activating physiological responses to counteract either an increase or decrease in heat loss, as well as heat production. However, major increases or decreases in environmental temperatures may be too great for the adaptive powers of the thermoregulatory center. The hypothalamic temperature regulating mechanisms have been shown to be impaired at temperatures below 34.5 °C, and begin to fail entirely at 29.5 °C (Guyton, 1986). On occasion, the surgical patient may undergo sufficient body cooling to prohibit any self-regulation of temperature. The function of the thermoregulatory center is also depressed by the administration of general anesthesia (Flacke et al., 1983), furthering impairing the patient's ability to regulate body temperature.

Mechanisms of Heat Loss

The human body can lose heat with the external environment via four methods. These are: radiation,

conduction, convection, and evaporation (Jensen, 1980). Radiant heat loss occurs when thermal energy is exchanged between two objects that are not in contact with each other and possess different temperatures. Heat is lost from warmer to cooler objects and the rate of loss is dependent on the temperature difference (Jensen, 1980). The cool environment of the operating room decreases the temperature of objects within the room. Heat from the patient is then lost to these cooler objects.

Conductive heat loss occurs as a result of direct contact between two objects possessing different temperatures. The transfer of heat is from molecule to molecule and moves down a thermal gradient (Vander et al., 1980). Thus, an object in direct contact with a cooler object will transfer heat to the cooler object.

Loss of body heat secondary to convection occurs when the skin temperature is warmer than the external environment. Heat is transferred from the skin to the surrounding air. This air mass becomes warmer, and then rises to be replaced by cooler air. The process is then repeated in a cyclic manner (Vander et al., 1980).

The final method of heat exchange is evaporative heat loss. Evaporative heat loss occurs when thermal energy is used to transform water from the liquid to the gaseous state. The heat used to fuel the process on the body surface is then lost via conduction. In humans, evaporative

heat loss occurs mainly via the skin and mucous membranes (Jensen, 1980). Evaporative heat loss is further increased when the protective integrity of the skin is compromised as occurs with the majority of surgical procedures.

Hypothermia

Normothermia is defined as a core body temperature of 36.7 to 37.6 °C. Hypothermia is defined as a cooling of the body's core temperature, and most authors agree that a core temperature of 35.0 °C or less constitutes hypothermia (Martyn, 1981). Hypothermia is commonly divided into three categories according to measured core temperature (a) mild (i.e. 33-35 °C), (b) moderate (i.e. 30-33 °C), and (c) severe (i.e. < 30 °C) (Lonning, Skulberg, & Abyholm, 1986).

With mild hypothermia, the patient shivers extensively to increase heat production. Heat production occurs as a result of increased muscle activity caused by shivering (i.e. rapid, involuntary, and random muscle contractions). In order for muscle fibers to contract, an enzyme located in muscle fibers, adenosine triphosphatase, must split adenosine triphosphate (ATP). ATP is a high energy compound which binds the actin-myosin cross bridge of the muscle fiber. The splitting of the ATP allows for movement of the cross bridge (muscle contraction). This action results in the release of energy in the form of heat, and formation of adenosine diphosphate (ADP). The resulting ADP must be

converted back to ATP for future energy needs via processes that require oxygen (e.g. oxidative phosphorylation). Thus, shivering increases heat production as well as oxygen consumption (Vander et al., 1980).

During mild hypothermia, sympathetic activity rises resulting in peripheral vasoconstriction that minimizes heat loss. The cardiac output, heart rate, and respiratory rate are increased to augment oxygen delivery to the tissues, this is secondary to the increased metabolic demand due to shivering. Peripheral vasoconstriction reflexively increases central circulation, augmenting renal blood flow which promotes a "cold diuresis". Depressed enzyme activity within the kidney is resposnsibe for the cold diuresis. The enzyme depression is a direct effect of the cold. This enzyme depression decreases reabsorption at the distal tubule leading to a loss of fluid and vital electrolytes (Coniam, 1979). If this diuresis is not corrected, dehydration and an increased hematocrit can occur within a period of several hours. A raised hematocrit will increase systemic vascular resistance, placing increasing blood viscosity, which puts increased demands on the heart to maintain cardiac output (Lonning, et al., 1986). Hemoconcentration also causes an increase in blood viscosity. Increased blood viscosity decreases microcirculatory blood flow due to increased resistance. As the microcirculatory blood flow decreases, the chances for

the development of metabolic acidosis as cellular metabolic byproducts accumulate (Rupp & Severinghaus, 1986).

In moderate hypothermia, shivering gradually decreases, followed by joint and muscle stiffness. The individual's level of consciousness is decreased due to the depressant effects of cooling on the central nervous system (Lonning et al., 1986). Cardiac output, heart rate, and blood pressure also begin to fall. These effects further decrease the body's ability to combat decreasing core temperature. Decreases in body temperature also cause a conformational change in the hemoglobin molecule causing oxygen to bind tightly. Subsequently, the oxygen-hemoglobin disassociation curve is shifted to the left, impairing the release of oxygen from hemoglobin at the tissue level (Martyn, 1981). Pathological electrocardiogram (ECG) changes begin to appear in the patient suffering moderate hypothermia. The ECG changes appear to be a direct result of cold on the sino-arterial node and atrio-ventricular node (Tolman & Cohen, 1970).

With severe hypothermia, most of the pathophysiologic conditions appearing in moderate hypothermia will appear in an exaggerated form. The ECG deteriorates to ventricular fibrillation and asystole, respiratory rate is decreased to gasps, bronchial secretions are increased secondary to decreased ciliary function, and respiratory and metabolic acidosis occurs (Lonning et al., 1986).

Pathophysiology of Hypothermia

Cellular pathophysiology. Humans suffering from hypothermia exhibit a variable physiologic response which is dependent upon the duration and severity of the temperature drop. Martyn (1981) points out that changes in human cells exposed to cold stress, in conjunction with changes mediated by the thermoregulatory center, are responsible for many of the pathological features seen in hypothermia. These changes include:

1. Cell membrane dysfunction resulting in impaired transportation of cellular nutrients and metabolites that eventually lead to cell death.

2. Cellular dehydration causing toxic concentrations of intracellular enzymes and electrolytes which disrupt normal cellular function.

3. A decrease in the rate oxygen disassociates from hemoglobin leading to anoxia, cellular infarction, and cellular death. Adequate oxygen delivery is vital for cellular function. All cells utilize oxygen in the mitochordria to synthesize ATP, which is then used to fuel cellular metabolism (Vander et al., 1980).

The cellular response to hypothermia is highly complex and subsequently involves all tissues and organ systems. The degree of organ dysfunction is dependent upon the severity and length of exposure to the stresses of cold.

Electrocardiogram. In early hypothermia (core temperature down to 33 °C), the heart rate is increased secondary to sympathetic stimulation. With progressive hypothermia, heart rate falls and eventually leads to junctional rhythms, atrial flutter, premature ventricular contractions, ventricular fibrillation, and asystole (Orkin & Cooperman, 1983). ECG changes seen with progressive hypothermia include prolonged P-R intervals, widening of the QRS complexes, prolongation of the Q-T intervals, ST segment elevation, and the characteristic "Osborn" wave occurring at temperatures below 31.0 °C. The "Osborn" wave is an extra, acute elevation of the ST segment. These dysrhythmias and ECG changes are the result of the temperature's depressant effect on the sino-arterial node, in addition to the slowing of depolarization and repolarization within cardiac cells (Orkin & Cooperman, 1983).

Cardiovascular. The initial increase in heart rate (HR) in early hypothermia increases cardiac output (CO) and mean arterial pressure (MAP). Peripheral vasoconstriction results in a relative increase in central blood volume. The increase in HR and peripheral vasoconstriction is accompanied by elevated catecholamines, primarily norepinephrine. As hypothermia progresses, CO and MAP decrease with the HR while peripheral vasoconstriction continues (D. Morris, Chambers, M. Morris, & Sande, 1985). The decrease in CO and increasing vasoconstriction, along

with the hemoconcentration that occurs with hypothermia, are responsible for the reduction in regional blood flow to the skeletal muscles, extremities, splanchnic circulation, and peripheral microcirculation. This decrease in blood flow predisposes the patient to metabolic acidosis and peripheral microvascular collapse. Increases in regional metabolic by-products and coagulation of stagnant blood are primarily responsible for these effects (Rupp & Severinghaus, 1986).

Cerebral function. For each degree Celsius drop in body temperature, cerebral blood flow and metabolic rate decrease six to seven percent (Martyn, 1981). As a result, the atriovenous oxygen difference is unchanged and cerebral hypoxia does not occur. However, depression of all central nervous system function is seen (Rupp & Severinghaus, 1986). Sedation occurs with mild hypothermia followed by changes in mentation and impaired motor function. Below 31.0 °C, there is a progressive paralysis of the central nervous system with unconsciousness developing below 29.0 °C. Cold induced depression of brain tissue metabolism is primarily responsible for these changes (Coniam, 1979).

Kidney. Renal function is reversibly depressed during hypothermia because of decreased systemic blood pressure and the direct effect of the cold temperatures (Orkin & Cooperman, 1983). The cold temperatures depress enzyme activity at the distal renal tubule resulting in decreased reabsorption and secretion. The ability of the kidney to

concentrate or dilute urine is impaired. The shift of fluid from the peripheral to the central circulation and the kidney's failure to carry out tubular reabsorption result in the "cold diuresis" that is observed in early hypothermia (Coniam, 1979).

Liver. Splanchnic blood flow and hepatic function decrease proportionately with body temperature. Therefore, the liver's ability to detoxify and conjugate drugs administered during general anesthesia is impaired. The CNS, cardiovascular, and respiratory depressant effects of many anesthetic drugs will persist for longer periods of time in the presence of hypothermia. This results in a slower emergence from general anesthesia by the patient, placing them at increased risk for complications. In the presence of hypothermia, the coagulation time of blood is increased secondary to depressed fibrinogen activity and the sequestration of platelets within the liver (Rupp & Severinghaus, 1986). This places the patient at risk for increased blood loss and hemodynamic compromise during surgery.

Pulmonary. The initial respiratory response to hypothermia is stimulation, followed by depression in proportion to the fall in body temperature and rate of metabolism. A decreased ventilatory response to increased carbon dioxide and decreased oxygen tension also occurs. The respiratory control center, located in the medulla, is

progressively depressed as body temperature decreases (Coniam, 1979). Spontaneous respiration normally ceases when the body temperature reaches 24.0 °C. In addition to the changes in respiratory control, there are changes in respiratory mechanics. Physiologic dead space, the part of the tidal ventilation that is unavailable for gas exchange, and anatomic dead space, the section of the respiratory tract that does not participate in gas exchange, are increased as a result of cold induced bronchial dilation. This results in a smaller portion of the tidal ventilation being available for oxygen exchange with the lung (Orkin & Cooperman, 1983).

Hypothermic patients emerging from anesthesia shiver to restore body heat. Shivering increases oxygen consumption up to 450% (Kaplan, 1983). Following anesthesia, the changes in pulmonary function induced by hypothermia include (a) increased dead space, (b) decreased respiratory response to carbon dioxide and, (c) a shift of the oxyhemoglobin curve to the left. These changes result in a decrease in the patient's ability to adequately oxygenate tissues, and thus places the patient at increased risk for developing hypoxia and possible ischemia of the myocardium.

Anesthesia and Hypothermia

It is common knowledge today that a profound decrease in patient temperature may occur under anesthesia unless

specific measures are undertaken to decrease heat loss. Factors that influence the rate of heat loss experienced by a patient include the age of the patient, temperature of the operating theater, type of operation, anesthetic agents used, infusion of cold intravenous fluids, and the use of cold dry anesthetic gases (Hall, 1978).

Infants, young children, and elderly adults are more likely to develop hypothermia than young and middle aged adults. Infants and young children have a larger body surface area to weight ratio, low basal metabolism rate, and imperfect sweating mechanism that predisposes them to hypothermia, especially under anesthesia (Hey, 1972). At the other extreme, elderly adults have a decreased ability to produce heat, and thus a compromised ability to increase heat production in the face of heat loss (Goldberg & Roe, 1966).

The environment of the operating theater is another factor predisposing the patient to hypothermia. Normally the temperature is maintained at 20 to 22 °C for the comfort of the surgical team. The cool temperature of the operating theater significantly increases heat loss from the patient via conductive and convective mechanisms (Hall, 1978).

It has also been shown that the type of operation performed has a significant effect on the amount of heat lost by the patient. Operations involving the abdominal and pleural cavities have been shown to increase the amount of

heat loss more than surgical procedures not involving body cavities (Morris, 1971). Surgeries involving skin grafting from large donor sites are associated with increased amounts of heat loss. This finding is due to large areas of the body's surface losing the ability to attenuate heat loss.

The use of anesthetic agents interfere with the normal thermoregulatory mechanisms of the patient and place them at greater risk for developing hypothermia. Many of the intravenous agents depress brain activity and consequently the central thermoregulatory center. Therefore, normal mechanisms to counteract hypothermia are lost. The use of neuromuscular blocking agents paralyze muscle activity, decreasing the heat derived from shivering. Potent inhalational agents produce peripheral vasodilation resulting in greater heat loss from the periphery, as well as preventing vasoconstrictive measures to conserve heat (Hall, 1978).

Inhalation of cold, dry anesthetic gases is another cause of heat loss in the surgical patient. Upon inhalation, the anesthetic gases must be warmed and humidified by the lungs which consumes a significant amount of body heat (Stone, Downs, Paul, & Perkins, 1981). Patients lose 7.8 ml of water vapor per hour warming and humidifying gases delivered through a standard semiclosed circle anesthesia circuit. This correlates to a 5.2 kilocalories per hour (kcal/hr) heat loss, which represents

7.6% of basal heat production (Graff & Benson, 1968). Endotracheal intubation further exacerbates this effect as the upper airway, that normally provides much of the warmth and humidity, is by-passed.

During surgical procedures, large quantities of fluids and blood can be administered to the patient. This includes fluid replacement for (a) preoperative fluid deficits, (b) intraoperative maintenance fluids, (c) intraoperative evaporative fluid loss, and (d) intraoperative blood loss. Finally, the administration of intravenous fluids at room temperature causes a reduction in total body heat as the heat of the body is used to warm the incoming intravenous fluids (Hall, 1978).

Post-Operative Complications of Hypothermia

The heat lost by the patient during anesthesia must be regained in the post-operative period. Shivering and vasoconstriction are often seen as compensatory mechanisms. However shivering can increase oxygen consumption by a value equal to 130 to 450% over normal levels (Kaplan, 1983). The increase in oxygen consumption occurs at a time when the patient is compromised in their ability to increase oxygen delivery secondary to the residual effects of anesthesia. Increasing the depth and rate of breathing in the surgical patient may result in increased levels of pain. The increased pain may act as a deterrent for the patient to

compensate with adequate breathing patterns. This problem can have serious consequences for the patient who has compromised cardiovascular or pulmonary functions (Arieff & Defronzo, 1985).

Prevention of Hypothermia

During the intraoperative period, the anesthetist is responsible for maintaining the patient in a normothermic state and preventing the deleterious effects of hypothermia. Numerous methods have been employed in order to conserve heat in the surgical patient. These methods include (a) wrapping the extremities with warm towels, (b) increasing the temperature of the operating theater, (c) humidification and warming of inhaled gases, (d) the use of heating blankets, (e) warming antiseptic and irrigation solutions, and (f) the warming of blood and intravenous fluids prior to administration. As stated by Boyan and Howland (1964), the warming of blood and fluids prior to administration is probably the most effective means of preventing hypothermia.

Blood warmers. Maintaining normothermia in the surgical patient is an important responsibility of the anesthetist. It is well known that the deleterious effects of hypothermia can be precipitated following the infusion of cold blood in the anesthetized patient. Blood is normally delivered to the operating room directly from the refrigerator. It is therefore necessary for the anesthetist

to warm the blood prior to infusion to prevent a drop in the patient's temperature.

The most common method utilized today for warming blood is the warm water bath method. In this particular warmer, the cold blood is infused through a coil that is submersed in an electrically heated water bath. The temperature of the bath is maintained at or near 37.0 °C. Blood is then warmed by the process of conduction. It then travels via output tubing from the warmer to the patient. In order for the warmer to be effective, it must deliver blood to the patient at a temperature of at least 32.0 °C (Russell, 1974). Temperatures above 45.0 °C however cause red cell hemolysis (Wilson & Iserson, 1987). A drawback reported by several researchers investigating the effectiveness of blood warmers concerns the amount of heat loss via the output tubing at low infusion rates (Russell, 1969; Vaghadia, 1984).

Mechanisms of Heat Exchange in the Blood Warmer

The major factors affecting heat exchange in the warm water bath blood warming devices are flow rate, length of intravenous tubing, the conductivity of the plastic intravenous tubing, and the temperature gradient between the water bath and the blood. Manipulation of these four variables can either increase or decrease the effectiveness of the blood warmer.

In a warm water bath blood warmer, heat is transferred to the blood via conduction through the tube wall and the layers of blood. With relatively slow infusion rates the blood moves via a process known as laminar flow. This flow is a property of fluid dynamics. The property states that fluids in a tube move via distinct and separate layers. The layers are parallel to each other and to the walls of the tubing. The velocity of flow is less in the layers at the periphery of the tubing. As you progress to the center of the tubing, the velocity of the layers progressively increase (Klein, 1984). Since blood behaves with all the properties of a fluid despite its many solid components, the diameter of the tubing through which it flows will affect its flow properties. As the diameter increases, the velocity of the inner fluid layers increases. Therefore, less conductive heat exchange occurs within the warmer. In the presence of laminar flow, blood layers remain constant and the diameter of the tubing becomes the major variable in conductive heat exchange (Russell, 1969).

The length of the blood warmer output tubing in combination with the flow rate determines the amount of time the warmed blood is in contact with the cool ambient air. Due to the temperature gradient, the warmed blood will lose heat to the cooler air via convection. Slow flow rates and/or longer output tubing will therefore increase the amount of heat loss between the blood warmer and the

patient. In order to decrease heat loss in the warmer output tubing, Russell (1969), Linko & Palosaari (1979), and Vaghadia, (1984) have suggested using the shortest length of tubing possible as well as insulating the output tubing. The researchers postulated that convective and conductive heat loss would be diminished with output tubing insulation.

Summary

Hypothermia is a complication of anesthesia that places the patient at risk to develop numerous pathophysiological sequelae. The heat debt that occurs during anesthesia must be repaid post-operatively and subsequently places the patient at further risk. The hypothermic state decreases liver blood flow and may prolong the serum half lives of many of the intravenous anesthetic drugs. It is important that the anesthetist employ whatever means available to preserve heat and prevent hypothermia.

Chapter Two

Review of Literature

Boyan and Howland (1961) published the first landmark studies regarding the beneficial effects of warming blood prior to transfusion. With the use of a thermocouple placed in the esophagus, measurement of the patient's body temperatures receiving massive blood transfusions were obtained. The researchers compared the body temperatures of patients receiving large amounts of unwarmed blood to those receiving warmed blood. It was found that in patients receiving large quantities of unwarmed blood (i.e. 4,000 to 18,000 ml), esophageal temperatures decreased to as low as 27.5 °C. Changes in cardiac function were associated with this drop in esophageal temperatures. EKG changes included premature ventricular contractions, bradycardia, ventricular fibrillation, and asystole. Boyan and Howland concluded that the drop in body temperature and changes in cardiac function were related to the patient's body mass, speed of transfusion, temperature of the stored blood, and the length of time the patient was exposed to the cool ambient air of the operating theater.

To study the effect of an infusion of warmed blood, Boyan and Howland (1961) developed an in-line blood warmer. The warmer consisted of a 24 foot length of plastic tubing, 4.5 millimeters in diameter, coiled and immersed in a 20 liter water bath maintained at 37.0 °C. In laboratory tests, the warmer was found to deliver blood at temperatures between 33.0 to 35.0 °C at flow rates between 50 and 100 ml/min. In clinical studies conducted on three patients receiving between 7,200 and 7,800 ml of blood at an average rate of 60 ml/min, the esophageal temperature remained within one degree of pre-transfusion levels and no cardiac abnormalities were noted. In patients whose esophageal temperatures had dropped after receiving cold blood, the warmer was effective in reversing the hypothermia and returning the patient's temperatures to normal limits. The researchers concluded that hypothermia precipitated by the transfusion of cold banked blood, could be prevented by warming the blood prior to transfusion.

In reporting the findings of their study, Boyan and Howland failed to cite the number of patients utilized in the study. No attempt was made to control room temperature, or the temperature of the banked blood prior to infusion or warming. No controls were placed on the type of surgery, length of surgery, or age of patients. These early findings, though lacking in methodology, did illustrate the need to warm blood prior to infusion.

Subsequent studies by Boyan and Howland (1963, 1964) utilizing the blood warmer on surgical patients yielded similar results. In the 1963 study, patients undergoing radical surgery for cancer were divided into two groups. Group one consisted of 45 patients who received 3,000 ml or more of warmed blood. Group two consisted of 36 patients who received 3,000 ml or more of unwarmed blood. A comparison of intraoperative cardiovascular changes between the two groups was profound. It was found that patients receiving large amounts of warmed blood were "warm, dry, and pink". All had readily obtainable blood pressure and pulse measurements and, as a group, suffered less cardiac disturbances than those receiving unwarmed blood. In addition, the group receiving warmed blood regained consciousness more readily than patients receiving cold blood. In the 1964 study, Boyan and Howland used the same methodology and placed 118 patients in the group receiving warm blood. The investigators compared the cardiovascular changes in this group to those seen in patients receiving cold blood from their 1963 study and again, similar results were found. It was their conclusion that definite beneficial effects were derived from the infusion of warmed blood versus the infusion of cold blood.

Boyan and Howland did show an improvement in methodology in their 1963 and 1964 studies by controlling for specific types of surgery and amounts of blood

administered. The patients studied underwent radical cancer surgery receiving 3,000 ml or more of blood. No mention was made of operating room temperatures, the length of surgical time, or other methods to prevent heat loss by the patient, nor were specific patient temperatures reported.

Morris and Trachtenberg (1968) studied the effects of cold versus warmed blood infusions in 12 patients requiring moderate amounts of blood replacement for major, nonvascular surgery. Patients requiring between 1,500 to 5,000 ml of blood were divided into two six member groups. One group received cold blood while the other group received blood passed through a coil immersed in a water bath kept at a constant temperature of 39.0 °C. The mean delivery temperature for the group receiving cold blood was 15.0 °C. The mean delivery temperature was 33.0 °C for the group receiving warmed blood. Utilizing a Swan Ganz cather, arterial line, and esophageal temperature probe, the changes in esophageal temperatures, cardiovascular function, and acid-base balance between the two groups were then compared. Morris and Trachtenberg reported four significant differences between the groups:

1) Esophageal temperatures decreased an average of 0.4 °C in the group receiving cold blood, while esophageal temperatures increased 0.4 °C in the group receiving warmed blood.

2) Mean cardiac output (calculated from a dye dilution curve) increased 6.6% in the group receiving cold blood infusions and increased 50% in the group receiving warmed blood infusions.

3) Mean peripheral vascular resistance (as calculated from the cardiac output, mean arterial pressure, and right atrial pressure) increased 4% in the cold blood group and was decreased 28% in the group receiving warmed blood.

4) Mean arterial carbon dioxide tension decreased 4% in the group receiving cold blood contrasted to a 13% increase in the group receiving warmed blood.

Morris and Trachtenberg (1968) believed that the measured differences between the groups was due to stimulation of the hypothalamic thermoregulatory control center by the warmed blood, rather than the small increase or decrease in measured core temperature. This stimulation resulted in a decrease in peripheral vascular resistance and an increase in cardiac output, thus improving cardiac performance.

Morris and Trachtenberg (1968) failed to mention whether operating room temperature was controlled and if any measures were taken to prevent intraoperative heat loss by the patients (i.e., covering with warm blankets; amounts of irrigation solutions used). The researchers also neglected to correlate patient temperatures closely with the administration of blood. The only patient temperatures

reported were the control measurements after induction of anesthesia. Subsequent temperatures were reported to have remained within 0.4 °C of the control for the duration of the study.

A study by Copping, Mather, and Winkler in 1971, compared the physiologic responses observed after the administration of cold, warm, and room temperature fluids in three groups of anesthetized dogs. The dogs were allowed to freely hemorrhage until their mean arterial pressures measured 30 to 40 mm of mercury. The investigators then administered Ringer's lactate solution based on four times the red cell mass lost plus 1.3 times the plasma volume lost. Group one (five dogs) received cold fluid (4.4 °C), group two (6 dogs) received room temperature fluid (21 to 27 °C), and group three (5 dogs) received warmed fluid (33 to 35 °C).

Blood pressure, pulse, ECG configurations, and esophageal temperatures were recorded at the beginning of the experiment, after hemorrhage, and during infusion of the various fluids. Three of five dogs died in the cold fluid infusion group while no deaths occurred in the dogs receiving room temperature infusions or warmed fluid infusions. Bradycardia and rhythm disturbances were seen in all dogs receiving cold fluids. Dogs in the other two groups demonstrated no rhythm disturbances and no statistically significant change in pulse rate. Blood

pressure responded poorly to fluid resuscitation in the cold fluid infusion group. Room temperature and warmed fluid infusions produced a significantly improved blood pressure response. Esophageal temperatures fell an average of 7 degrees Fahrenheit (°F) in the cold fluid infusion group, 4 °F in the room temperature infusion group, and 0.6 °F in the group of dogs receiving warmed intravenous fluids. Copping, Mather, and Winkler (1971) concluded that their study confirmed the deleterious effects of infusing large amounts of unwarmed fluids.

Copping, Mather, and Winkler (1971) did not mention the environmental temperature when the study was performed. Fluid temperatures were only monitored at the start of infusion, no controls were implemented to keep the fluid temperature constant. The researchers also failed to mention whether any cooling or warming of the fluids occurred during the infusion process secondary to environmental temperature. Also there was a failure to specify in the tables of data whether the blood pressures monitored were MAP or systolic pressures.

In a discussion of warmed fluid heat exchangers, Russell (1969) states that the warming and subsequent infusion of blood can be analyzed theoretically. The author further discusses the variables one should consider when designing a water bath blood warmer. Russell (1969) recommends:

1) "The warmer tubing should be as large as possible to minimize the flow resistance within the limits of an acceptable priming volume.

2) The tubing should allow for maximum heat exchange within the warmer.

3) The output tubing should allow for maximum heat conservation between the warmer and patient.

4) When infusing at low flow rates, the use of short, insulated output tubing is recommended to minimize heat loss to the environment." (p. 345)

Russell (1969) designed and tested a water bath blood warmer based on these recommendations. This warmer was tested for heat exchange efficiency in an in vitro laboratory trial. Sterile ice water maintained at a temperature of 0-2 °C was used for the experiment. The environmental temperature was maintained at 20.0 °C. Temperatures of the infusing sterile water were monitored at the entry point of the warmer, in the water bath (maintained between 39.5 to 40.5 °C), at the exit point of the warmer, and at the point of patient entry. An electric thermometer was used to monitor the water bath temperature, while a thermistor was used to measure temperature at the other points. All thermometers used in the study were calibrated against a mercury thermometer. Testing showed that a significant amount of heat loss occurred in the output line at flow rates less than 20 ml/min. Russell's data showed

that heat loss from the output tubing averaged 3.6 °C at a flow rate of 24 ml/min, 8.9 °C at 9.7 ml/min, and 11.1 °C at 6.4 ml/min.

A problem with Russell's study concerned the control of flow rate. He relied on gravity flow with a roller wedge controller to control flow rate. The roller wedge controller does not allow for precise control of flow and introduces variability of flow into the study.

Xifaras and Healy (1971) performed an in vitro study comparing the efficiency of disposable blood warming coils from four different manufactuers. The coils were compared with respect to the flow resistance, the temperature rise induced at various flow rates and, their clinical convenience. The researchers utilized a constant head of pressure (140 centimeters of water) to measure differences in resistance to flow. Immersing the coils in a warm water bath that was maintained at a temperature of 36.0 to 38.0 °C, ice water was passed through the coils. The input temperature at the proximal end of the coil, the water bath temperature, and the output temperature at the infusion site were measured at ten second intervals for several flow rates. Xifaras and Healy found that the temperature rise between the input and output of the coil depends on the rate of flow. It was also determined that ambient room temperature had a marked effect on the transfusion temperature. At low flows, considerable warming was found

to take place between the cold reservoir and the inlet of the warming coil. At the other extreme, blood temperature emerging from the coil was greater than room temperature. Therefore, the researchers concluded that a drop in temperature between the coil and the patient would occur.

A weakness in this study was the failure of the researchers to measure the temperature of the fluid as it exited the warmer. With such a measurement, a more precise calculation of heat loss via the output tubing could be made. Xifaras and Healy (1971) also failed to mention if the environmental temperature was kept constant during the study.

Linko and Palosaari (1979) investigated the warming of blood in a water bath, and cooling of blood at room temperature. Utilizing outdated blood, the investigators warmed the units of blood by three different approaches and compared the efficiency of these techniques. The three warming techniques used were (a) immersion of the blood units in a bucket of water warmed to 39.5 °C; (b) immersion of the blood units in a stirred, thermostat controlled water bath and; (c) immersion of the blood units in a thermostat controlled water bath with continuous agitation of the blood unit. The influence of blood flow on cooling was also studied. Six units of blood were warmed to a temperature of 37.0 °C, while the room temperature was kept constant at 21.0 °C. Temperatures were monitored within the blood

units, the transfusion set, and at the distal end of the output tubing while the blood flowed at rates of 50, 100, and 150 ml/min.

Linko and Palosaari (1979) found that at room temperature, the blood temperature decreased approximately 1.5 °C at a flow of 150 ml/min. At a flow rate of 100 ml/min, the blood temperature decreased 1.8 °C. Finally, at a flow rate of 50 ml/min, the temperature decreased approximately 3.0 °C. The conclusions were obvious, warmed blood cooled in the outlet tubing of the transfusion set. Furthermore, the degree of cooling depended on the flow rate used (i.e., increased flow rates equals decreased heat loss). Recognizing that cooling in the outlet tubing can be significant, the researchers recommended insulating the output tubing to prevent heat loss.

The methodology utilized by Linko and Palosaari was relatively stringent. The researchers used two different types of thermometers that were calibrated against a mercury thermometer to insure accuracy. Room temperature was maintained at 21.0 °C. Controlling room temperature during this aspect of the study was of extreme importance since ambient temperature is known to affect conductive and convective heat losses of the warmed blood.

Norman, Ahmad, and Zieg (1986) studied the changes in temperature of warmed versus room temperature infusions of lactated Ringer's solution using varying lengths of

intravenous tubing. One liter bags of lactated Ringer's were warmed to either 44.0, 50.0, or 60.0 °C, and the solution was infused at 500 or 1,000 ml/hr. A room temperature of 23 to 25 °C was maintained throughout the study. Temperatures were recorded at the following locations (a) the surface of the fluid container, (b) the inside the fluid container, (c) the connection to the blood administration set, (d) the proximal end of the extension set, and (e) the distal end of the extension set. Temperature measurements were recorded one minute after initiation of flow and at five minute intervals thereafter up to 1 hour. One liter containers of lactated Ringer's, at room temperature, were also infused in the same manner to serve as the control group.

During the first 5 minutes of the infusion, Norman et al. (1986) found that the warming of fluids resulted in an increase in temperature at the point of patient entry secondary to the heating of the intravenous tubing. Thereafter, a decline in temperature was noted which was attributed to heat loss from the tubing to the surrounding environment. It was also noted that an increase in delivery temperature could be achieved if the length of the intravenous tubing were shortened. The researchers found that there was an average heat loss of 19.2 °C when using tubing having a length of 195 centimeters (cm) with an 80 cm extension set. Temperatures monitored at the end of the 195

cm intravenous tubing was approximately 1.5 °C higher that those measured at the end of the attached 80 cm extension set. It was also concluded that increases in delivery temperature could be achieved if the bag and/or intravenous tubing was insulated, thus preventing heat loss to the environment.

Norman et al. (1986) utilized a Mon-A-Therm® temperature probe terminal with a three channel adapter and tympanic temperature probes to control for variation in temperature measurements. The investigators failed to mention whether room temperature was monitored or controlled. Since fluctuations in the environmental temperature could effect the results, a certain amount of doubt is cast upon the validity of their study.

Skrivanek and Hein (1986) evaluated the effect of various blood warmer output tubing lengths with regard to the delivery temperature of warmed Plasmalyte at the point of patient entry. Plasmalyte is a non-blood derived starch solution used to replace lost blood plasma. One liter bags of plasmalyte, warmed via a Fenwall blood warmer, were infused at flow rates of 18, 40, 60, 100, and 200 ml/min. Output tubing 1.2 meters (m) in length, output tubing with two 20 inch extension sets (total length 2.3 m), and output tubing with two 20 inch extension sets wrapped with aluminum foil (total length 2.3 m) were used in the study.

Temperatures were recorded within the room, the warmer, the plasmalyte, and the patient entry point.

Skrivanek and Hein (1986) found that at flow rates of approximately one liter per hour, fluid warming was found to be minimally effective in preventig heat debt (i.e. 4.1 k/cal per hour). Heat debt refers to the amount of energy the body must expend to maintain normothermia when a decrease in body temperature occurs. Normal body heat production is one k/cal per hour (Stone, Downs, Paul, & Perkins, 1981). With higher flow rates, fluid warming was found to be more effective, preventing a heat debt of 58 k/cal per hour at 100 ml/min. In the normal adult, this would decrease the energy expenditure to maintain normothermia by 50%.

Increased output tubing length was found to increase conductive and convective heat loss from the warm fluid. Wrapping the tubing in aluminum foil was ineffective in reducing heat loss. It was concluded that fluid warming is most effective at flow rates between 40 and 100 ml/min.

Skrivanek and Hein (1986) used aluminum foil as an insulative material to evaluate its effect on decreasing heat loss from intravenous tubing. Aluminum foil, while effective in preventing reflective heat loss is not effective in preventing convective and conductive heat. It is then logical to investigate the efficiency of insulative

materials in preventing convective and conductive heat loss from intravenous tubing.

Vaghadia (1986) investigated flow rates and delivery temperature in 23 patients receiving blood warmed by disposable blood warming devices. With flow rates less than 30 ml/min, delivery temperatures to the patients were slightly greater than the ambient operating room temperature (i.e., 0-2 °C). Vaghadia suggested that when infusing fluids at a rate less than 30 ml/min, consideration should be given to insulating the blood warmer output tubing to prevent heat loss. No formal investigation was undertaken by Vaghadia to validate his suggestion.

Baker (1985) and Cooter (1987) conducted <u>in vitro</u> studies that correlated flow rates to the amount of heat lost between the exit point, and point of patient entry, in Hemokinotherm and Fenwall® blood warmers. Methodology for the two studies were very similar except Baker diluted each unit of outdated blood with 100 cc of normal saline prior to warming. Cooter placed the units of blood in an ice bath and then infused the units through a blood warmer. Flow rates, regulated with a volumetric infusion pump, ranged from 50 to 4,000 ml/hr in Baker's study and 100 to 8,000 ml/hr in Cooter's study. The temperature of the operating room was maintained at 20.0 to 21.0 °C. Temperatures were recorded within the ice bath containing the unwarmed units

of blood, at the entry point of the blood warmer, at the exit point of the blood warmer, and at the point of patient entry.

At flow rates below 400-500 ml/hr, Baker (1985) found a net temperature loss of 8.7 °C between the exit point of the blood warmer and the point of patient entry. At higher flow rates (i.e. between 500 and 3,000 ml/hr), the temperature was found to progressively decrease (7 °C at 600 ml/hr, 2.8 °C at 3,000 ml/hr). The author concluded that the difference in delivery temperatures was due to the varying exposure time of the warmed blood in the output tubing to the cool ambient air. It was also concluded that the routine use of blood warmers should be questioned when flow rates are below 400 to 500 ml/hr.

Cooter (1987) obtained similar results using the Fenwall blood warmer. At flow rates between 100 and 600 ml/hr, the temperature of the blood at patient entry was, at the greatest, only 2 °C above the operating room temperature. Cooter's data showed a decrease in blood temperature between the blood warmer and patient at all flow rates investigated. The largest decrease occurred at the lower infusion rates. At an infusion rate of 100 ml/hr, a decrease in temperature of 9.2 °C was found. Infusion rates of 7,992 ml/hr showed a decrease in temperature of 2.0 °C.

Rymers (1988) duplicated Cooter's study while manipulating the length of the blood warmer output tubing.

Rymers' data showed the peak efficiency of the Hemokinatherm® blood warmer occurred at flow rates between 400 and 3,000 ml/hr. The efficiency of the warmer at flow rates below 400 ml/hr decreased. The author concluded that heat was lost by conduction and convection as the blood spent more time in the output tubing. Rymers observed a mean heat loss of 7.97 °C in the output tubing at flow rates between 100 to 3,000 ml/hr. At flow rates between 3,100 to 7,000 ml/hr, a mean heat loss of 4.750 °C was observed. The study also showed that the temperature of the blood at patient entry decreased as the length of the output increased. A decrease in temperature of approximately 0.8 °C occurred with the addition of a 33 inch extension tubing. This loss was again attributed to greater amounts of convective and conductive heat loss to the environment.

Rymers' (1988) results were consistent with those of Baker (1985), Cooter (1987), Skrivanek and Hein (1986), and Vaghadia (1986). Each researcher found a significant heat loss occurring in the output tubing after fluids or blood had been warmed. Two conclusions can be made as a result of these studies: (a) there is an inverse relationship between flow rate and heat loss in the blood warmer output tubing (i.e., as flow rate decreases heat loss increases); (b) there is a direct relationship between output tubing length

and heat loss in the blood warmer output tubing (i.e., as output tubing length increases heat loss increases).

Summary

Earlier studies support the belief that infusion of cold fluids and blood products can lower body temperature and precipitate alterations in normal physiological function. These changes can increase the morbidity and mortality of patients undergoing general anesthesia and surgical procedures. The warming of fluids and blood products prior to infusion is a common practice utilized by anesthetists in an attempt to maintain normothermia in the surgical patient. The literature is quite clear in support of the concept that heat loss occurs in the blood warmer output tubing. The studies show that heat gained from the warmer is lost from the output tubing via conductive and convective mechanisms. This heat loss can be quite dramatic and render the process of fluid warming ineffective at low flow rates. Some investigators believe that insulation of the output tubing could decrease this heat loss, but few have studied this recommendation. Consequently, an investigation was proposed and implemented to provide the answer to this question.

Chapter Three

Methodology

Design

In an attempt to learn whether the application of insulation to blood warmer output tubing is effective in decreasing heat loss from warmed packed red blood cells at flow rates less than 1,200 ml/hr, a true experimental design was used. A control group, consisting of non-insulated blood warmer output tubing and an experimental group, with insulated blood warmer output tubing were examined. Since significant heat loss has been reported to occur in non-insulated blood warmer output tubing at flow rates less than 1,200 ml/hr, flow rates of 300, 600, and 900 ml/hr were studied.

Population and Sample

The population consisted of all Fenwall blood warming coils. The sample for the study was obtained from the main operating room at a Mid-Atlantic, university based, metropolitan medical center. A total of two warming coils were selected for the study.

Instrumentation

DUPACO-Hemokinatherm® Blood Warmer (model #32400). The Hemokinatherm® blood warmer is an in-line blood warmer with an automatic system for warming cold blood during intravenous infusions. The warmer contains a 600 ml water basin which is warmed via an electrically powered warmer. During use, warming coils are submerged within the basin. The blood enters the coils where it is warmed to a temperature range of 30.0 to 37.0 °C. (DUPACO Incorporated, 1740 LaCosta Meadows Drive, San Marcos, California 92069.)

Mon-A-Therm® Model 6500 Temperature Monitoring System. The Mon-A-Therm® Model 6500, two channel, temperature monitoring system was utilized to determine the blood's temperature at specific points. This system can measure temperatures from 1.0-50.0 °C with an accuracy of ± 0.1 °C. The system has a pre-set calibration with an automatic check and is shielded against radio frequency interference. (Mon-A-Therm® Incorporated, 520 South Jefferson Avenue, St. Louis, Missouri 63103.)

Infusion pump. A Travenol Flo-Gard 8000 volumetric infusion pump, which can deliver from 1 to 999 ml/hr depending on the flow rate chosen, was used to control blood flow rates. The pump is reported to be accurate at 125 ± 4 ml/hr by the manufacturer. (Travenol Laboratories, 1 Baxter Parkway, Deerfield, Illinois 60015).

Warming coil. The Fenwall Laboratories blood warming coil with an output tubing extension 69 inches in length was used for the study. This is a high density polyethylene intravenous tubing with a 54.0 ml capacity. (Fenwall Laboratories, Division of Fenwall Laboratories Incorporated, Deerfield, Illinois 60015.)

Three Bentley C-125 disposable plastic connectors with an inside diameter of 0.25 inches with a luer lock adapter were used. The connectors were attached in-line to the blood warming coils at the entry point of the blood warmer, the exit point of the blood warmer, and the end of the output tubing (point of patient entry). This allowed for connection of the in-line temperature probes. (American Bentley, Subsidiary of American Hospital Supply Corporation, 17502 Armstrong Avenue, Irvine California 92714)

Infusion tubing. One Travenol Lab Volumetric Pump Administration Set was used. The set was connected to the blood unit and the entry point of the blood warming coil to allow for use of the infusion pump. (Catalog number 2C1031, Travenol Laboratories, 1 Baxter Parkway, Deerfield, Illinois 60015)

Temperature probes. Four Mon-A-Therm® luer lock temperature sensors were used in the study. Three probes were used to monitor the temperature of the packed red blood cells at the entry point of the blood warmer, the exit point

of the blood warmer, and the end of the output tubing. Also, one sensor was used to monitor the temperature of the room. (Catalog number 503-0501, Mon-A-Therm Incorporated, St. Louis, Missouri 63103.)

Output tubing insulation. Macklanburg-Duncan foam weatherstrip tape, measuring 0.5 inches thick and 0.75 inches wide, was used to insulate the blood warmer output tubing. (Macklanburg-Duncan, Oklahoma City, Oklahoma 73118.)

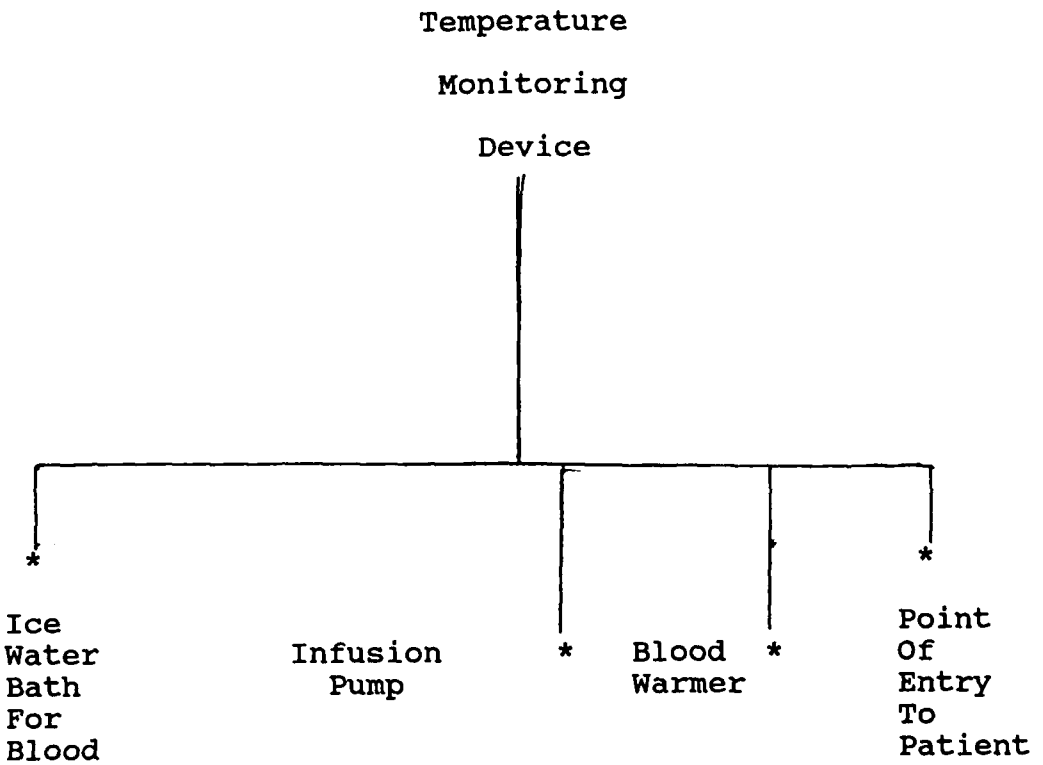
Procedures

The control and experimental groups were compared under artificial conditions within an operating room at the medical center. This room is typical of an operating room at any major hospital. All attempts were made to replicate a true surgical setting. The temperature of the operating room was maintained at 20.0 $\pm$ 0.5 °C. All lights that would normally be used during an actual surgical procedure were illuminated. The Bentley disposable plastic connectors, used to connect the temperature probes, were attached in-line to the blood warming set at three points (a) the entry point to the blood warmer, (b) the exit point of the blood warmer, and (c) the point of patient entry. The temperature probes were then attached to the connectors via the luer lock connection, and in turn connected to the temperature monitor.

The equipment and instruments were arranged as outlined in Figure 1. The Hemokinatherm® blood warmer was filled with sterile water, according to the manufacturer's specifications and placed on the floor near the head of the operating room table. The water bath was allowed to warm for a period of 15 min. Undiluted, outdated packed red blood cells were cooled to a temperature of 0-1 °C. The volumetric pump tubing was attached to the red blood cell units. The tubing was then inserted in the volumetric pump. Tubing from the infusion pump was connected to the blood warming coil which was placed in the Hemokinatherm® blood warmer. Packed red blood cell (PRBC) temperatures were recorded as the PRBCs entered the blood warmer, as the PRBCs exited the blood warmer, and at the point of patient entry. The point of patient entry is the exit point of the pre-attached extension tubing that comes with the blood warming coils.

After priming the pump tubing and warming coil with blood, the infusion was started. After the initiation of flow, the temperature of the PRBCs were recorded at 10 minute time intervals. Temperatures were recorded at these intervals for a period of 30 minutes at each of the points specified in Figure 1. Output tubing wrapped with the described insulation and non insulated output tubing were

Figure 1. Equipment setup diagram



Note. Points of temperature measurements are indicated by *.

compared. Flow rates of 300, 600, and 900 ml/hr were investigated in both groups. The study was performed twice to verify accuracy of the results.

Statistical Analysis

Statistical analysis for the effect of insulating blood warmer output tubing on the temperature of packed red blood cells at low flow rates was examined by an analysis of covariance, using a three by three by two factorial experiment with replicates. The three by three by two factorial experiment allowed for analysis of the three levels of flow rate, three levels of time in the warmer or output tubing, and two levels of insulation (with and without). The dependent variable was the blood temperature as it enters the patient, and the covariate was blood temperature as it left the warmer.

The data was first tested for "parallelism" by examining the interaction between the treatment variable and the covariate. Parallelism (homogeneity of slopes) is an important assumption underlying the analysis of covariance. With this assumption, the magnitude of any contrast among the treatments' means at one value of the covariate will equal the magnitude of that contrast at any other. In order to run an analysis of covariance, parallelism between the treatment variable and covariate must be shown. Analyses were performed using SYSTAT, a statistical software package

(SYSTAT, 1988). The particular routine used was the multivariate general linear hypothesis module.

Chapter Four

Results

The hypothesis was tested using a true experimental design. The purpose of the experiment was to determine what effect insulating blood warmer output tubing had on the temperature of warmed packed red blood cells at low flow rates. The control group consisted of non-insulated output tubing and the experimental group consisted of insulated output tubing. Independent variables included insulated output tubing and low flow rates (i.e. 300, 600, and 900 ml/hr). The dependent variable was the amount of heat loss that occurred in the output tubing. Room temperature was maintained at 20.0 $\pm$ 0.6 °C.

The raw data obtained from this study are presented in tabular form in the Appendix. The effect of low flow rates on heat loss in non-insulated and insulated blood warming output tubing are presented in Tables 1 and 2. Examination of the data shows that mean heat loss decreased as flow rate increased in both insulated and non-insulated blood warmer output tubing. In the non-insulated, heat loss decreased 3.8 °C when flows were increased from 300 to 600 ml/hr

Table 1

Flow Rate Effect On Heat Loss

Flow rate (ml/hr)	Heat loss* (°C)	
	Non-insulated**	Insulated**
300	12.017 ± 0.391	8.617 ± 0.091
600	8.217 ± 0.164	6.600 ± 0.186
900	6.617 ± 0.105	3.883 ± 0.098

* Mean temperature ± the standard error of the mean

** Blood warmer output tubing

Table 2

Insulative Effect On Output Tubing Heat Loss

Flow rate (ml/hr)	Mean heat preservation* (°C)
300	3.400
600	1.617
900	2.734

* Insulated versus non-insulated output tubing

and 1.6 °C when increased from 600 to 900 ml/hr in the non-insulated group. In the insulated group, heat loss decreased 2.0 °C when flows were increased from 300 to 600 ml/hr and 2.7 °C when increased from 600 to 900 ml/hr. Further examination revealed the mean heat preservation decreased as flow rate increased from 300 to 600 ml/hr and then increased as flow rate increased from 600 to 900 ml/hr in insulated blood warmer output tubing. Analysis of the data showed a statistically significant difference between heat loss in non-insulated versus insulated output tubing at low flow rates.

Insulated versus non-insulated output tubing.

The effect of insulated versus non-insulated blood warmer output tubing on heat loss at low flow rates was examined by analysis of covariance, using a three by three by two factorial experiment with replicates. The data was first tested for parallelism by examining the interaction between the treatment variable and the covariate. The test for parallelism revealed an F of 1.462 which corresponds to a p of 0.235. The p is not significant at an alpha level of 0.05, the hypothesis of parallelism is not rejected and an analysis of covariance is justified.

Using an analysis of covariance, the data revealed a statistically significant decrease in heat loss using insulated output tubing. The usual alpha level of 0.05 was

adjusted to 0.005 because of the large number of effects being analyzed (Bonferroni correction for multiple comparisons). This decreases the risk of basing conclusions on data measurements obtained by random chance. The effects of flow and insulation revealed a p of 0.011. Thus, statistical analysis showed that insulation of the blood warmer output tubing significantly increased the temperature of blood entering the patient as compared to non-insulated tubing.

Chapter 5

Discussion

The purpose of this true experimental study was to determine what effect insulating the blood warmer output tubing had on the temperature of packed red blood cells at low flow rates. Results obtained from the study showed that a statistically significant difference in heat loss occurred between the insulated and non-insulated groups. Therefore, the null hypothesis which stated there would be no statistical difference in heat loss between insulated and non-insulated blood warmer output tubing at flow rates below 1200 ml/hr was rejected.

Correlation With Previous Studies

The results obtained in the control group study (non-insulated output tubing) support to the findings of previous studies (Russell, 1969; Linko and Palosaari, 1979; Vaghadia, 1986; Baker, 1985; Cooter, 1987; Rymers, 1988). These researchers found that significant heat loss occurs in the blood warmer output tubing. Results showing heat loss from non-insulated output tubing averaged 12.0 °C at a flow rate

of 300 ml/hr and 8.2 °C at 600 ml/hr correlated well with Russell's findings. Russell reported average heat losses of 11.1 °C at a flow rate of 384 ml/hr and 9.7 °C at 582 ml/hr. Linko and Palosaari (1979) obtained similar results reporting an average heat loss of 3.0 °C with flow rates of 3000 ml/hr.

Findings differed somewhat from those reported by Vaghadia (1986). Vaghadia reported that at flow rates of less than 1800 ml/hr, delivery temperatures of warmed blood averaged zero to only two °C above room temperature. The results of this study found that delivery temperatures ranged from 2.4 °C above room temperature at 300 ml/hr to 8.0 °C at 900 ml/hr. The differences in these findings may be due to the differing methodologies utilized in the studies. In this particular study, the end of the output tubing was secured at the head of the operating room table and was not in contact with the floor or any other object within the room. Vaghadia fails to mention in his study whether these precautions were taken. If not, a significant amount of heat loss would occur as the blood exits the warmer and enters the area of coldest room air near the floor. Also contact with other cold objects within the room would increase the amount of conductive and convective heat losses that occur in blood warmer output tubing.

Similar arguments can be made for the differences in blood delivery temperatures as reported by Cooter (1987).

Cooter found that with flow rates between 100 and 600 ml/hr, blood delivery temperatures were only two degrees above ambient room temperature.

Baker (1985) reported heat losses in blood warmer output tubing similar to those found in this study. At flow rates between 400-500 ml/hr, heat loss averaged 8.7 °C. Baker also reported a steady decrease in heat loss as flow rate increased. These findings are similar to the results of this study.

Effect of Insulation on Heat Loss

Insulation of blood warmer output tubing significantly decreased the amount of heat loss that occurred between the exit point of the blood warmer and the point of patient entry in comparison to non-insulated tubing. Analysis of covariance revealed a $p < 0.001$ (at an alpha level of 0.005) when comparing insulated to non-insulated blood warmer output tubing on the temperature of warmed blood at the point of patient entry. Results revealed that insulation, on the average, decreased heat loss 3.4 °C at a flow rate of 300 ml/hr, 1.6 °C at 600 ml/hr, and 2.7 °C at 900 ml/hr. The rationale for this affect is that the insulative material decreased the amount of conductive and convective heat losses that occur in non-insulated output tubing.

Effect of Flow Rate and Insulation on Heat Loss

Statistical analysis showed that flow rate had a significant effect on the amount of heat loss that occurred in the blood warmer output tubing. Analysis of covariance revealed a $p < 0.001$, at an alpha level of 0.005, when analyzing the effect of flow rate on the temperature of warmed blood at the point of patient entry. Flow rate was found to inversely affect heat loss. In other words, the higher the flow rate, the warmer the blood entering the patient. As flow rates increased form 300 to 900 ml/hr, heat loss decreased 5.4 °C in the non-insulated group and 4.7 °C in the insulated group. This seems reasonable, inasmuch as the blood spends less time in the output tubing and therefore loses less heat through conductive and convective mechanisms.

An interesting finding in this study was the interaction between flow and insulation. While there was a strong interaction between flow in the non-insulated group ($p = 0.000$), the interaction was weak in the insulated group ($p = 0.011$). The only explanation for this result is that the insulative material's ability to decrease heat loss nullified the effect of increased flow rates seen in the non-insulated group.

Another interesting finding in the insulated group, was the drop in efficiency that occurred at the 600 ml/hr flow rate. Initially, changes from laminar to turbulent flow was

thought to be the reason. A change from laminar to turbulent flow in fluids occurs as flow rate increases. Laminar flow produces a slower moving layer of blood near the tube wall. Turbulent flow causes increased mixing of the blood. This results in greater amounts of convective and conductive heat losses, as larger amounts of warm blood are in contact with the sides of the tubing. The explanation of turbulent flow was discarded because the decreased efficiency was not observed at the 900 ml/hr flow rate. Subsequently, no plausible explanation for this finding could be found.

Difficulties With the Study

The only difficulty encountered in the study was the inability to explain the decreased efficiency of insulated output tubing at the 600 ml/hr flow rate.

Recommendations for Further Study

1. Numerous surgical procedures require the administration of intravenous fluids at the studied flow rates, thus replicate the study using intravenous fluids instead of packed red blood cells.

2. Replicate the study using different types of insulative materials to compare their effectiveness.

Conclusions

The hypothesis was rejected as analysis indicated a statistically significant difference existed in heat loss between insulated and non-insulated blood warmer output tubing at low flow rates. On the basis of the data obtained, the following conclusions can be made (a) insulating the blood warmer output tubing significantly decreases the heat loss that occurs between the warmer and the point of patient entry, and (b) lower flow rates result in an increased heat loss in both non-insulated and insulated blood warmer output tubing.

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Appendix

Appendix

Results

Note. T-1 = room temperature

T-2 = temperature at entry point of blood warmer

T-3 = temperature at exit point of blood warmer

T-4 = temperature at point of patient entry

* all temperaures measured in °C

Non-insulated Output Tubing, First Run

Flow Rate (ml/hr)	Minutes	T-1	T-2	T-3	T-4
300	10	20.5	19.8	34.7	23.1
300	20	20.6	19.8	34.8	23.7
300	30	20.6	19.2	34.8	23.9
600	10	20.6	17.4	35.2	26.9
600	20	20.5	18.1	35.3	27.0
600	30	20.6	17.7	35.2	26.3
900	10	20.5	15.9	35.2	28.5
900	20	20.5	15.6	35.1	28.1
900	30	20.5	15.0	35.1	28.7

Non-insulated Output Tubing, Second Run

Flow Rate (ml/hr)	Minutes	T-1	T-2	T-3	T-4
300	10	20.1	18.8	34.8	21.9
300	20	20.3	18.8	34.8	22.4
300	30	20.3	19.4	35.2	22.0
600	10	20.3	16.5	33.7	25.6
600	20	20.2	16.9	33.7	26.0
600	30	20.2	17.2	34.0	26.0
900	10	20.1	15.8	34.5	27.7
900	20	20.1	16.4	34.6	28.2
900	30	20.0	17.4	34.6	28.2

Insulated Ouptut Tubing, First Run

Flow Rate (ml/hr)	Minutes	T-1	T-2	T-3	T-4
300	10	20.5	19.7	34.3	25.6
300	20	20.5	19.9	34.4	26.1
300	30	20.5	20.2	34.3	25.8
600	10	20.5	18.9	34.5	27.2
600	20	20.5	19.1	34.1	27.5
600	30	20.5	19.5	34.0	27.5
900	10	20.5	15.0	34.4	30.3
900	20	20.5	15.6	34.2	30.5
900	30	20.5	15.6	33.8	30.3

Insulated Output Tubing, Second Run

Flow Rate (ml/hr)	Minutes	T-1	T-2	T-3	T-4
300	10	20.0	19.0	33.4	24.5
300	20	20.0	19.0	33.2	24.4
300	30	20.0	19.2	33.2	24.7
600	10	20.0	17.1	33.9	27.1
600	20	20.3	17.4	33.8	27.3
600	30	20.3	17.8	34.1	28.2
900	10	20.4	16.8	34.2	30.1
900	20	20.4	17.2	33.8	29.9
900	30	20.3	17.6	34.0	30.0

Vita

James Peter Holt [redacted]. [redacted], and is an American citizen. He graduated from Oak Ridge High School, Oak Ridge, Tennessee in 1974. He received a Bachelor of Arts in Zoology from the University of Tennessee, Knoxville, Tennessee in 1979. He later earned a Masters of Science in Nursing from the University of Tennessee, Knoxville, Tennessee, in 1983. Following graduation, he was commissioned as an officer in the United States Air Force where he holds the present rank of Captain. He is married to the former [redacted] and they have two children, [redacted]. He returned to school in August, 1988, in the Department of Nurse Anesthesia, School of Allied Health Professions, Virginia Commonwealth University.

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This is to certify that the thesis prepared by James Peter Holt entitled THE EFFECT OF INSULATING BLOOD WARMER OUTPUT TUBING ON THE TEMPERATURE OF PACKED RED BLOOD CELLS AT LOW FLOW RATES has been approved by his committee as satisfactory completion of the thesis requirement for the degree of Master of Science in Nurse Anesthesia.

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